



				38770 S CONTOUR			
PubMed	Nucleotide	Protein	Genome	Structure	PopSet	Taxonomy	OMIM
Search PubMe	d ▼ for					Go Clear	
· · · · · · · · · · · · · · · · · · ·	L	_imits	Preview/Index	History	Clipbo	ard	
	*						
	Displi	ay Abstract	▼ Save	Text Ord	der Addito	Clipboard	
Entrez PubMed	d						

□ 1: *J Immunol* 1995 May 15;154(10):5590-600

Related Articles, Books, LinkOut

PubMed Services

Administration of noncytolytic IL-10/Fc in murine models of lipopolysaccharide-induced septic shock and allogeneic islet transplantation.

Zheng XX, Steele AW, Nickerson PW, Steurer W, Steiger J, Strom TB

Harvard Medical School, Department of Medicine, Boston, MA, USA.

Related Resources

Numerous studies have suggested the potential application of IL-10 as an anti-inflammatory and as an antirejection agent. Unfortunately, cytokines have short circulating t1/2 We developed a murine IL-10/Fc gamma 2a immunoligand that possesses the biologic functions of IL-10 and the long circulating t1/2 in vivo, characteristic of Igs. We mutated the Fc gamma 2a fragment to render the immunoligand ineffective in directing Ab-dependent cell-mediated cytotoxicity and complement-directed cytolysis (noncytolytic IL-10/Fc (IL-10/Fc2-)). In terms of IL-10 activity, IL-10/Fc2- was as effective as rIL-10 mole per mole in preventing lethal septic shock, but the immunoligand had a prolonged period of efficacy in accord with its extended circulating half-life. Contrary to expectations, IL-10/Fc2- treatment tended to accelerate the destruction of islet cell allografts and increase the levels of granzyme B gene expression in local draining lymph nodes. These data suggest that the enhanced cytotoxic activity of allograft-destroying CTLs may contribute to the accelerated allograft rejection. Finally, our studies suggest that a noncytolytic IL-10/Fc fusion protein provides a useful tool to study the biologic effects of IL-10 in vivo and may provide a useful agent for the prevention and treatment of septic shock.

PMID: 7730658, UI: 95248127

Add to Clipboard Save Text Order Display Abstract

> Write to the Help Desk NCBI | NLM | NIH Department of Health & Human Services Freedom of Information Act | Disclaimer







PubMed	Nucleotide	Protein	Genome	Structure	PopSet	Taxonomy	OMIM					
Search PubM	led ▼ for					Go Clear						
	l	Limits	Preview/Index	History	Clipbo	ard						
	*:											
	Displ	ay Abstract	▼ Save	Text On	der Addito	Clipboard						
Entrez PubMe	ed		.,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,									

☐1: Cancer Res 1999 Jun 15;59(12):2924-30

Related Articles, Books, LinkOut

PubMed Services

Differential effects of a stem cell factor-immunoglobulin fusion protein on malignant and normal hematopoietic cells.

Erben U, Thiel E, Notter M

Department of Hematology, Oncology and Transfusion Medicine, Universitaetsklinikum Benjamin Franklin, Berlin, Germany.

Related Resources

We genetically connected the extracellular domain of human stem cell factor to the Fc-portion of human IgG1. The chimeric recombinant stem cell factor IgG1 fusion protein (rSCF-IgG1) had an apparent approximately Mr 190,000 and consisted of three identical covalently linked subunits. It specifically bound to c-kit and the high affinity Fc gamma receptor, respectively. Liquid phase rSCF-IgG1 was, on a molar basis, about eight times more potent than native human rSCF in stimulating the proliferation of c-kit-positive leukemic cell lines and of nonmalignant CD34-positive hematopoietic progenitor cells. Although the effective dose conferring half maximum of [methyl-3H]thymidine uptake by liquid phase and solid phase-bound rSCF-IgG1 were comparable, the plateau level of [methyl-3H]thymidine uptake by malignant cells was decreased by the latter, whereas proliferation of nonmalignant progenitor cells was supported. Liquid phase rSCF-IgG1 had a 2-fold increased potential to maintain primitive nonmalignant progenitor cells in stroma-free long-term culture compared with rSCF. Liquid phase rSCF-IgG1 caused enhanced and prolonged receptor phosphorylation and a more rapid down modulation of c-kit. Our data support the concept that solid phase-attachment of rSCF-IgG1 is sufficient for alteration of biological function and that rSCF-IgG1 partially blocks SCF-stimulated malignant cell growth while supporting normal progenitor cells.

PMID: 10383156, UI: 99310529

Display Abstract Save Text Order Add to Clipboard

Write to the Help Desk

NCBI | NLM | NIH

Department of Health & Human Services

Freedom of Information Act | Disclaimer